

REFERENCES

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REPLY

We appreciate the interest by Drs. Henrikson and Chandra-Strobo in our recent study (1). They disagree with the conclusion that lower levels of troponin are likely analytical false positives and are more likely related to necrosis. This is based on the results of their recent study (2), in which patients with minor troponin T (TnT) elevations had a higher event than those without detectable levels, and were closer to those with clearly elevated levels (2).

However, the two studies are not directly comparable. The range of TnT levels, which they called "marginal" (defined as levels between 0.01 ng/ml and 0.09 ng/ml), was fairly broad. The TnT levels from 0.01 to 0.03 ng/ml (between the lower limit of detectability and 10% coefficient of variation [CV]) (3) are equivalent to our low troponin I values, whereas those from 0.03 to 0.09 ng/ml (from the 10% CV to the prior myocardial infarction [MI] diagnostic criteria) would be equivalent to our intermediate TnI levels. The fact that their marginal TnT values are a combination of low and intermediate TnT values likely explains the higher event rate that was found. For example, the proportion of patients who had elevated creatine kinase-MB fraction (CK-MB) was 15% in our TnI intermediate group, comparable to 14% in their marginal TnT group; in contrast, only 1.1% of patients in our TnI low group had increased CK-MB.

In addition, we did not call patients with detectable TnI values "normal." We made the distinction between low elevations, in which some represent necrosis; this is seen by the higher event rate. However, as we and others have noted (4), this is a mixture of a small number of patients who truly have necrosis and a much higher number who have analytical false positives. As we noted, labeling a patient who has atypical chest pain, no-ischemic electrocardiographic changes, low levels of CK-MB not near the diagnostic cut-off, and with minor troponin elevations as having an myocardial infarction has significant implications for the patient's long-term health care, and we believe this is inappropriate. As none of the currently available assays conform to recommended standards (5), our data, as well as other recommendations (6), are that these values *may* be indicative of necrosis, and therefore should be further evaluated. This evaluation should be dependent on the clinical scenario. Rather than routinely performing coronary angiography as recommended by the American College of Cardiology/American Heart Association guidelines for pa-

tients who have troponin elevations, we believe that stress testing would be an appropriate evaluation, for many of these patients.

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The Diet-Heart Hypothesis: An Evolutionary Support

Nothing in biology makes sense except in the light of evolution.
Theodosius Dobzhansky, 1973

Considering that humankind and its metabolic physiology are biological products of evolution, one might well ask why Weinberg's report (1) not only ignores the evolutionary arguments supporting the diet-heart hypothesis, but also advances the evolutionarily untenable thesis that the low-fat, high-carbohydrate diet is responsible for the current epidemics of obesity, type II diabetes, and the metabolic syndrome.

Weinberg seems to forget that the low-fat, high-carbohydrate diet represents the diet "for which human beings are in essence genetically programmed" (2), because their metabolic physiology has been evolutionarily molded by a nutritional environment in which, for millions of years, that diet was practically the sole one available to our ancestors (3). For 99% of its evolution, humankind indeed lived mainly on fruits and vegetables, which consist essentially of carbohydrates, and consumed little fat, because game was very lean and cattle-breeding, chicken farming, butter, dairy products, margarines, and oils did not exist (2).

Weinberg fails to note that coronary heart disease and type II diabetes were virtually absent in some traditional populations that ate mainly carbohydrates and consumed two to three or even five times less fat than Westerners (2). As evidence that the diet-heart hypothesis is correct, those populations, after their switch to Western diets, are currently facing epidemics of cardiovascular disease (2), type II diabetes (2,3), and obesity (3).

Weinberg also fails to note that in rural China, where dietary energy derives mostly from carbohydrates and the consumption of fat is less than one-half that of Americans, coronary artery disease mortality is 16.7-fold lower than that in the U.S. (3). As an additional support of the diet-heart hypothesis, in urban China the proportion of cardiovascular disease deaths rose from 12.1% in 1957 to 35.8% in 1990 because of the greatly increased fat consumption, favored by the global availability of cheap vegetable oils (2).

Weinberg's critique of the diet-heart hypothesis is based on the misleading results of dietary trials, most of which investigated the effects of diets that were called "low-fat" inappropriately. As a paradigmatic example, if we consider that diets exceeding 10% to 15% of energy as fat were virtually impossible in the low-fat nutritional environment that shaped our metabolic physiology (3), the expression "low fat" is inappropriate to define a diet providing 31% of energy as fat (4). The beneficial effects of an evolutionarily defined low-fat diet providing only 10% of energy as fat are unquestionable (5).

Weinberg argues that low-fat, high-carbohydrate diets are unhealthy because they decrease high-density lipoprotein (HDL) cholesterol and increase triglycerides. However, populations that consume mostly carbohydrates, despite having low HDL cholesterol, are virtually free from coronary heart disease (2). Moreover, the low-fat, high-carbohydrate diet, instead of increasing triglycerides, may actually reduce them, as long as sugar intake is kept low (5), to limit the harmful effects caused by sugars consumed in solid forms or in solutions exceeding the physiologic limit imposed by evolution, namely 4.18 MJ/l (3).

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REPLY

In his discussion of my commentary on the diet-heart hypothesis (1), Dr. Baschetti suggests that the low-fat, high-carbohydrate diet is the diet "for which human beings are in essence genetically programmed, because their metabolic physiology has been evolutionarily molded by a nutritional environment in which, for millions of years, that diet was practically the sole one available to our ancestors." However, the references he cites for documentation are merely similar letters to editors in which he previously expressed the same opinions, a practice he has pursued beyond those cited here (2). I would point out to Dr. Baschetti that my conclusions about the role of the low-fat, high-carbohydrate diet in the current epidemics of obesity, type II diabetes, and metabolic syndrome are based on what has actually occurred in the U. S. and elsewhere over the last three or more decades. In my critique, I also cited several recent randomized trials in which low-carbohydrate, high-protein diets showed improvement in lipid patterns, insulin sensitivity, and weight loss, compared with low-fat, high-carbohydrate diets. Although Dr. Baschetti dismisses these studies as "misleading" they are well-controlled trials at highly regarded institutions, and they were performed by scientists who were highly circumspect in self-appraisal of their work.

Dr. Baschetti also argues, as he did in his cited letter to Dr. M. B. Katan (3), that rural Chinese on low-fat, high-carbohydrate regimens have less coronary disease than occurs in the U. S., although it is rising as fat intake increases. Dr. Baschetti cites this as further evidence favoring the low-fat, high-carbohydrate diet. I respond as did Dr. Katan (3), that there are many other factors which make comparison of rural Chinese with American populations difficult, including rising use of cigarettes in China, different levels of physical activity, and of sanitation, health care, and the types of fats available to the low-income Chinese, such as tropical oils, high in saturated fat.

Further, to advocate a low-fat, high-carbohydrate diet because that was the diet primitive peoples ate millions of years ago and to claim that we, therefore, are genetically endowed for that to be the ideal diet, and beyond that to disregard the negative experience with the low-fat, high-carbohydrate diet on the U.S. and other populations and to reject as "misleading" positive randomized research data favoring the low-carbohydrate, high-protein diet, seems fanciful indeed.